Methods We studied 8 patients, identified from our anticoagulation database, who had been previously established on warfarin, and then commenced azathioprine or mercaptopurine for inflammatory bowel disease (2), systemic lupus erythematosus (1), nephritic syndrome (1), Wegener's granulomatosis (1), polyarteritis nodosa (1), dermatomyositis (1) and renal transplant (1). The effect of thiopurine on international normalised ratio (INR), and warfarin dose prior to and following commencement of thiopurine was recorded.

Results In 6/8 patients, following introduction of azathioprine or mercaptopurine, the warfarin dose had to be significantly increased (100% [18–500], Median [range]) in order to maintain a therapeutic INR. Any subsequent reductions in thiopurine dose were mirrored by a rise in INR and lower requirement for warfarin.

In 2 IBD patients, each with a high warfarin requirement, thiopurine metabolites were measured. In both patients MeMP:TGN ratio was >11. Thiopurine dose was reduced to 25% and allopurinol 100 mg added. INR was carefully monitored. In both cases INR increased within a week (to 6.9 and 11.2) and warfarin doses were subsequently reduced by ½ and 2/3 respectively to regain therapeutic INR.

Conclusion It is important for clinicians to be aware of the potential inhibitory action of thiopurines on warfarin's anticoagulant effect. Close INR monitoring is essential when initiating thiopurines and especially when reducing their dose and/or adding allopurinol. Failure to recognise the latter could result in bleeding due to over-anticoagulation. The high MeMP:TGN ratio in 2 of our patients also raises the possibility that thiopurine metabolites may play a role in the interaction between thiopurines and warfarin.

Disclosure of Interest None Declared.

PWE-081 DIAGNOSTIC PERFORMANCE OF FAECAL CALPROTECTIN IN PRIMARY CARE

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Introduction Faecal calprotectin is recommended by NICE[1] for distinguishing between irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD) in patients with lower gastrointestinal (GI) symptoms in primary care. If cancer is suspected in these patients and 'red-flag' symptoms such as anaemia or bleeding, they should be referred to Gastroenterology in accordance with the NICE suspected cancer guideline.[2] We use calprotectin in secondary care and will be extending the service to primary care providers. However, a number of GPs have been requesting faecal calprotectin on an ad-hoc basis for 1 year, giving us valuable insight into how the test performs in primary care.

Methods An audit was carried out of primary care calprotectin data in a 1 year period (Dec 12–Dec 13). This data was compared to an audit of 1 month of secondary care data (Jun 13). Clinical details, such as endoscopy and histology results were extracted from electronic patient records.

Results In total 198 requests for calprotectin came from primary care in 1 year and 40 were unsuitable for analysis (wrong sample type or delayed arrival in lab). Of the remaining 158 calprotectin requests, 76% were considered appropriate, having clinical details including symptoms described by NICE. Worryingly, 17% of requests had inappropriate clinical details such as bleeding; such patients' referral to Gastroenterology was potentially delayed by requesting calprotectin. In 7% of requests no reason for request was discernable.

Of the primary care requests, 29% results were consistent with intestinal inflammation (>50 µg/g). If GPs use our proposed algorithm which suggests only referring patients with a calprotectin >50 µg/g, and those where strong clinical suspicion remains, there is potential for up to 71% reduction in patients referred to Gastroenterology with 'IBS/IBD' symptoms.

Diagnostic performance of calprotectin compared with endoscopy and histology diagnosis in secondary care is excellent with a sensitivity of 100% and a specificity of 91%. In primary care the corresponding data gives a sensitivity of 93% and a specificity of 79%.

Conclusion We received a large number of unsuitable samples. In addition GPs appear to be inappropriately requesting calprotectin in patients with symptoms such as bleeding, therefore it is critical to offer the service in a controlled way as part of a locally agreed care pathway. We are producing a GP information leaflet to advise on appropriate sample collection, result interpretation and the proposed patient pathway. We will re-audit primary care data once this is introduced to investigate whether a targeted approach leads to improved diagnostic performance of calprotectin in primary care.

REFERENCES

1 Faecal Calprotectin diagnostic tests for inflammatory diseases of the bowel, NICE DG11
2 Referral for suspected cancer, NICE CG27

Disclosure of Interest None Declared.

PWE-082 THE IMPACT OF NOD2 VARIANTS ON GUT MICROBIOTA IN CROHN'S DISEASE AND HEALTHY CONTROLS

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Introduction Crohn's disease (CD) is now understood to be caused by the interaction between genetic and environmental factors with dysregulation of gut microbiota playing a pivotal role. *NOD2*, the strongest genetic risk factor for CD, encodes a pattern recognition receptor and plays an important role in epithelial defence. Studies of *NOD2*-knockout mice have demonstrated shifts in gut microbiota. Human studies to date have been limited by relatively small numbers of individuals homozygous for *NOD2* mutations without accurate matching of controls.

Methods Individuals with CD of known *NOD2* status were identified from the UK IBD genetics consortium. Patients in clinical remission were selected if they carried 2 of the common *NOD2* variants (homozygotes or compound heterozygotes). Each *NOD2* mutant patient was matched to a *NOD2* wild-type